UNIVERSITY OF WISCONSIN-LA CROSSE

Graduate Studies

EFFECT OF DISPARITIES OF FEEDBACK ON PACING IN CYCLE TIME TRIALS

A Manuscript Style Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Science in Clinical Exercise Physiology

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ABSTRACT

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Introduction: The purpose of this study is to understand the effect of hypoxia during warm-up and competition on performance during cycle time trials. Methods: Seven well-trained subjects performed a VO_{2max} test, two habituation trials, and four randomly ordered, single-blind 5 km time trials. Subjects performed HH (hypoxic WU/hypoxic TT), HN (hypoxic WU/normoxic TT), NH (normoxic WU/hypoxic TT), or NN (normoxic WU/hypoxic TT) with hypoxia (F_iO_2 =0.15) and normoxia (F_iO_2=0.21). Results: The hypoxic warm-up elicited a significant (p<.05) decrease in S_aO_2 (hypoxic S_aO_2=86%, normoxic S_aO_2=97%) and increases in RPE and HLa. During the TT significant differences in PO between hypoxic and normoxic TT began at 2.0 km, continuing for the duration of performance (NN PO @ 1,2,3,4,5 km=271, 271, 260, 256, 304W. NH PO=251, 239, 219, 212, 247W. HN PO=259, 258, 257, 250, 294. HH PO=238, 215, 212, 205, 245). There was no significant difference in initial PO. Discussion: Despite manipulating the pre-exercise template, PO is not reduced until afferent physiological feedback occurs within the time trial, ~2.0 km. Apparently, with changes in F_iO_2 subjects cannot distinguish this change, even if a low F_iO_2 is presented during warm-up, until physiological feedback mechanisms have time to act during the time trial.

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INTRODUCTION

Pacing can be described as a strategy employed to avoid catastrophic failure of peripheral physiological systems (St Clair Gibson et al., 2006). There has been extensive interest in the area of pacing and the peripheral and central physiological mechanisms which control pacing. Athletes utilize pacing strategies for both competition and performance enhancement, and numerous practice hours are spent perfecting pacing strategy. However, pacing may also be described as a natural phenomenon of life and applicable to the overall functioning of our species, not unique to athletic competition (Noakes et al., 2005). Research examining this concept has included measurement parameters such as heart rate (HR), electromyography (EMG), power output (PO), and rating of perceived exertion (RPE) (Amann et al., 2006; Amann et al., 2007; Albertus et al., 2005; Ansley et al., 2004; Curry et al., In Press; Foster et al., 2003; Rauch et al., 2005; St Clair Gibson et al., 2001). Several models of pacing strategy have been presented, including models of both central and peripheral fatigue within a paced activity.

Swart et al. (2009) describes peripheral fatigue as "either the excessive accumulation or depletion of key chemicals which interferes with cross bridge cycling in the exercising muscles, impairing their capacity to produce force." Central fatigue "proposes that related chemical changes in the brain alter cerebral function, reducing central motor drive to the exercising muscles." The model of peripheral fatigue predicts that exercise always terminates at an absolute, temporarily irreversible end point, based on maximal muscle fiber recruitment and metabolic disturbance (St Clair Gibson &

Noakes, 2004). In an attempt to integrate central fatigue and peripheral fatigue Ulmer (1996) proposed the idea of "teleoanticipation" which involves a feedback control system including a programmer which anticipates and calculates an appropriate level of exertion with the finishing point taken into consideration. Teleoanticipation "includes both feed forward planning and feedback control from afferent nerves from peripheral metabolic structures and the external environment, and also incorporates knowledge acquired from prior exercise bouts" (Lambert et al., 2005). This concept of anticipatory strategies of pacing has been tested in numerous studies. Mauger et al. (2009) showed that distance feedback within a time trial is not necessarily advantageous to the development of a pacing strategy. This implies that a "central programmer" determines the appropriate level of exertion based on prior experience of similar activities, even before onset of the activity. St Clair Gibson et al. (2006) suggests that "the brain incorporates knowledge of the endpoint, memory of prior similar events, and knowledge of external (environmental) and internal (metabolic) conditions to set a particular optimal pacing strategy."

One environmental manipulator of pace is that of exercising in hypoxic conditions. Hypoxia may be particularly valuable as an experimental model to evaluate elements of pacing strategy since inspired gas concentrations can be manipulated while the subject is blinded. Noakes et al. (2001) suggested that the brain has the ability to sense acute hypoxia and to increase blood flow in response, and suggests that the regulation of motor unit recruitment is determined through calculations of the central programmer to prevent overexertion, which would jeopardize oxygen delivery to vital organs. Johnson et al. (2009) manipulated pacing strategies through a mid-event hypoxic challenge. The results of this study suggested that arterial oxygen saturation is an

important signaling mechanism regulating power output and implying the presence of central control through chemoreceptors. This information correlates with other studies discussing the idea that the brain and central nervous system (central governor) are in dynamic back and forth communication through afferent sensory pathways (Amann et al., 2006; Amann et al., 2007a; Foster et al., 2002; Foster et al., 2003; Lambert et al., 2005; St Clair Gibson & Noakes, 2004; St Clair Gibson et al., 2006). A recent study by Henslin et al. (In Press) suggested that arterial desaturation immediately prior to a time trial does not change the pattern of power output at the beginning of a time trial. These results call into question the findings of Johnson et al. (2009) that arterial desaturation can be directly sensed and is a direct controller of power output. These results suggest the importance of the pre-exercise template (which was not manipulated in the study of Henslin et al. (2009)) and the importance of changes in metabolic status which occur secondary to limitations in central oxygen transport following administration of hypoxia. The body must respond in such a way as to adapt to two independent stressors (hypoxia and exercise) therefore "the simultaneous presence of both stressors will have an additive effect that will influence maximal exercise capacity, endurance time to fatigue, and overall exercise performance" (Mazzeo, 2008).

The mechanisms behind the central programmer, afferent sensory pathways, and efferent response pathways are imperfectly understood. The purpose of this study was to further understand the components of pacing, that is, the relationship between anticipatory factors, previous experience, and physiological feedback mechanisms. Our question was in regard to the manipulation of the feedback template of exercise. If the athlete's pre-exercise template is altered by manipulating warm-up conditions will the

athlete's template change or will decreases in power output only occur when physiological feedback contradicts the anticipatory template. We hypothesize that when F_iO_2 changes from that expected during the warm-up, PO will adapt to match the PO appropriate for the F_iO_2.

MATERIALS AND METHODS

Subjects

The subjects were 7 well-trained cyclists familiar with time trial performance. This population was chosen based on familiarity with cycling time trial performances, since we want subjects with previous experience of the testing procedure. Written informed consent was obtained prior to beginning this study. Approval from the University of Wisconsin-La Crosse Institutional Review Board for the Protection of Human Subjects was obtained prior to the beginning of this study.

Table 1. Descriptive Statistics and Maximal Exercise Results.

Total (n = 7)		Age (years)	Mass (kg)	VO_{2max} (L/min)	HR_{max} (bpm)	PO_{max} (watts)
	Subject 1	21	65.91	3.30	178	300
	Subject 2	27	73.64	4.70	185	375
	Subject 3	23	62.84	3.34	184	275
	Subject 4	23	73.18	5.13	181	400
	Subject 5	33	75.00	4.87	172	350
	Subject 6	23	85.00	4.98	187	350
	Subject 7	33	70.00	3.94	159	325
	Average	26	72.22	4.32	178	339.29
	StDev	±5.01	±7.15	±0.78	±9.76	±42.96
Males (n = 5)						
	Average	28	75.36	4.72	177	360
	StDev	±5.02	±5.69	±0.47	±11.5	±28.5
Females (n = 2)						
	Average	22	64.38	3.32	181	287.5
	StDev	±1.41	±2.17	±0.03	±4.24	±17.68

Procedure

The subjects performed 7 exercise trials; one VO_2 maximal test performed on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands), two habituation trials (to control for learning effects), and 4 randomly ordered and single-blinded time trials on an electrically braked cycle ergometer (Velotron, Racermate, Seattle, WA). Mixing chamber based open circuit spirometry system was used to measure gas exchange (AEI Technologies, Naperville, IL).

Subjects completed the maximal oxygen consumption test to obtain exercise responses such as VO_{2max}, heart rate (HR), and power output (PO). Exercise intensity began at 25 W, and every 2 minutes was increased by 25 W until volitional fatigue.

Rating of perceived exertion (RPE) by the modified Borg CR-10 scale (Borg, 1998) and HR (Polar Vantage XL, Polar Instruments, Port Washington, NY) were recorded at the end of each stage. Measurements of 50% PPO (peak power output) and 75% PPO were recorded for the experimental warm-up stages.

Once this was complete subjects performed two habituation time trials to familiarize themselves with the testing procedures. Subjects performed 4 randomized, single-blinded time trials (TT) with either normoxic or hypoxic gas mixtures during the warm-up and competition phases. The trial conditions are as follows: hypoxic warm-up and hypoxic time trial (HH), normoxic warm-up and hypoxic time trial (NH), hypoxic warm-up and normoxic time trial (HN), and normoxic warm-up and normoxic time trial (NN). The normoxic gas mixture was ~21% oxygen (room air), while the hypoxic mixture was ~15% oxygen, (equivalent to 2300 meters altitude, the height of the highest Olympic competition). The selected gas concentration was breathed through a respiratory mask providing slight resistance for both hypoxic and normoxic breathing conditions, attempting to standardize both conditions.

Each warm-up phase consisted of 18 minutes, with the initial 5 minutes at 100 watts, 5 minutes at 50% of the subject's PPO (from the incremental test), 3 minutes at 75% of PPO, then 5 minutes at 100 watts. During this entire time, participants breathed the 'warm-up' gas mixture for that trial. At each incremental phase of the warm-up we recorded RPE, HR, blood lactate (HLa)(Accusport, Hawthorne, NY), and arterial oxygen saturation (S_aO_2). A fingertip pulse oximeter measure S_aO_2 throughout warm-up and TT conditions (Allegiance Oxi-Reader 2000, Allegiance Health Care, McGraw Park, IL). Once completed there was a 3 minutes transition time, where subjects moved from the

Lode cycle ergometer to the Velotron time trial bike and then cycled at 25 W until the TT start. The Desaturation Strain Index (HR*HLa*(100-S_aO_2)) was calculated for the warm-up conditions comparing normoxic and hypoxic measurements, indicating the amount of physiological strain experienced by the subject to provide evidence for a significantly altered template (Jaime et al., In Press).

The 'competition' gas mixture was administered 1 minute prior to the start of the time trial, yet participants were unaware of when this is occurring or which gas mixture they are receiving. We chose to administer the 'competition' gas mixture during this pre-exercise phase to ensure that arterial saturation was stable at the onset of exercise. Subjects completed a 5 km time trial, and every 500 meters we recorded RPE, HR, HLa, S_aO_2 and PO until the time trial was completed. Feedback was provided to the subject in regard to time, PO, velocity, HR, and distance completed.

Statistical Analysis

Repeated-measures ANOVA was used to determine the significance of changes in outcome variables. Comparisons were made of PO, HR, S_aO_2, HLa, RPE, and time for all warm-up and time trial conditions (HH, HN, NH, NN). Post hoc tests were performed when justified by ANOVA, using the Fisher's LSD. Statistical analysis of the warm-up conditions combined both hypoxic conditions against both normoxic conditions to find an average difference between the conditions. The same calculations were followed for analysis of the time trial conditions.

RESULTS

Significant differences were seen between the hypoxic and normoxic warm-up conditions. Illustrated in Figure 1 are the average power outputs for the duration of the performance, including the warm-up conditions. PO was regulated throughout the warm-up, but significant increases in blood lactate (HLa) ($p = .002$) and rate of perceived exertion (RPE) ($p = .044$) along with significant decreases in S_aO_2 ($p = .000$) were seen with the hypoxic warm-up condition, with no significant difference in HR (Figures 2-7). The Desaturation Strain Index (Jaime et al., In Press) showed significant differences between conditions ($p = .000$), suggesting that the physiological strain of performing in a hypoxic environment produced detrimental stress (Figure 8). Despite significantly different responses during the two warm-up conditions, there was no significant variation in the initial PO of the time trial. Since the warm-up was incrementally staged, PO was regulated according to the subject's peak power percentages (50% and 75%). Blood lactate (HLa) and RPE were all significantly greater in the hypoxic warm-up condition ($F_iO_2 = .15$) while S_aO_2 was significantly lower. The averaged results for the hypoxic time trials and the averaged results for the normoxic time trials were analyzed to compare the differences of conditions.

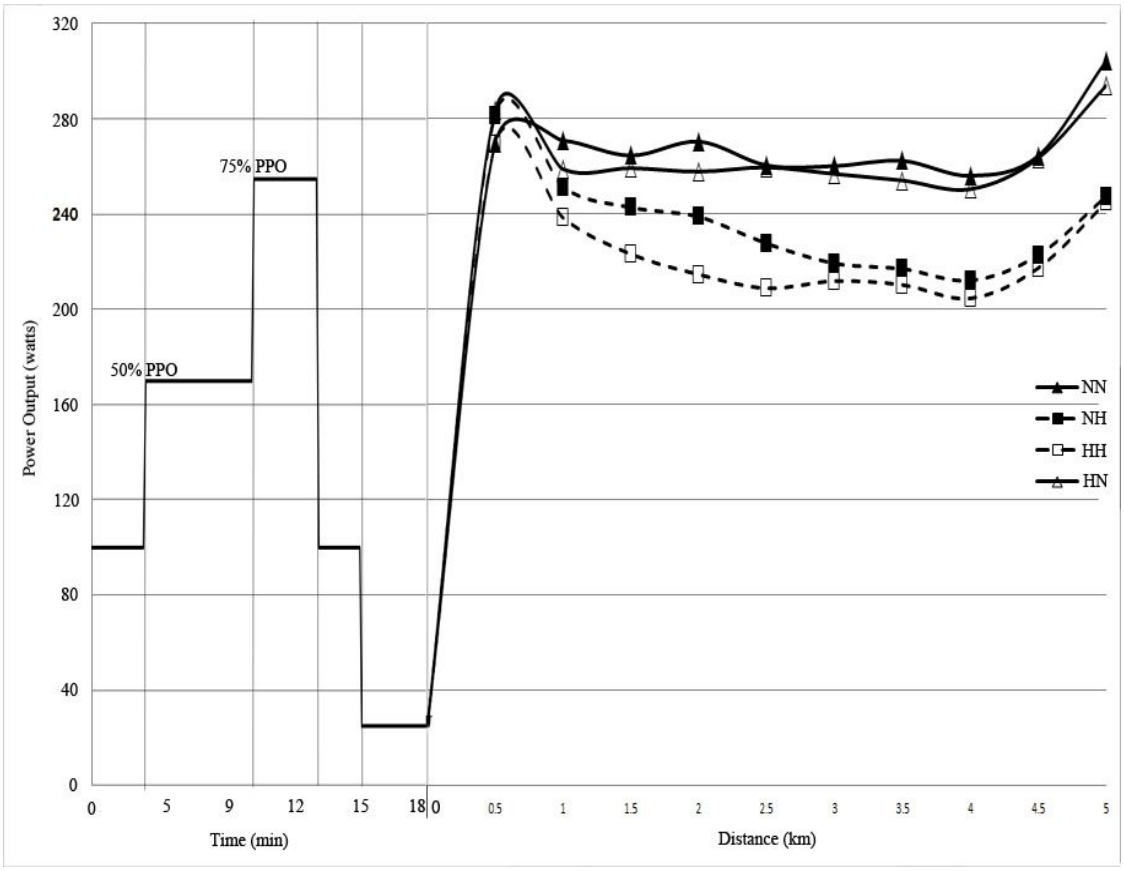

Figure 1. Average PO for all conditions for warm-up and time trial performances
*the x axis is altered from time (min) to distance (km) from the variability in completion time for the time trial with the given F_iO_2.

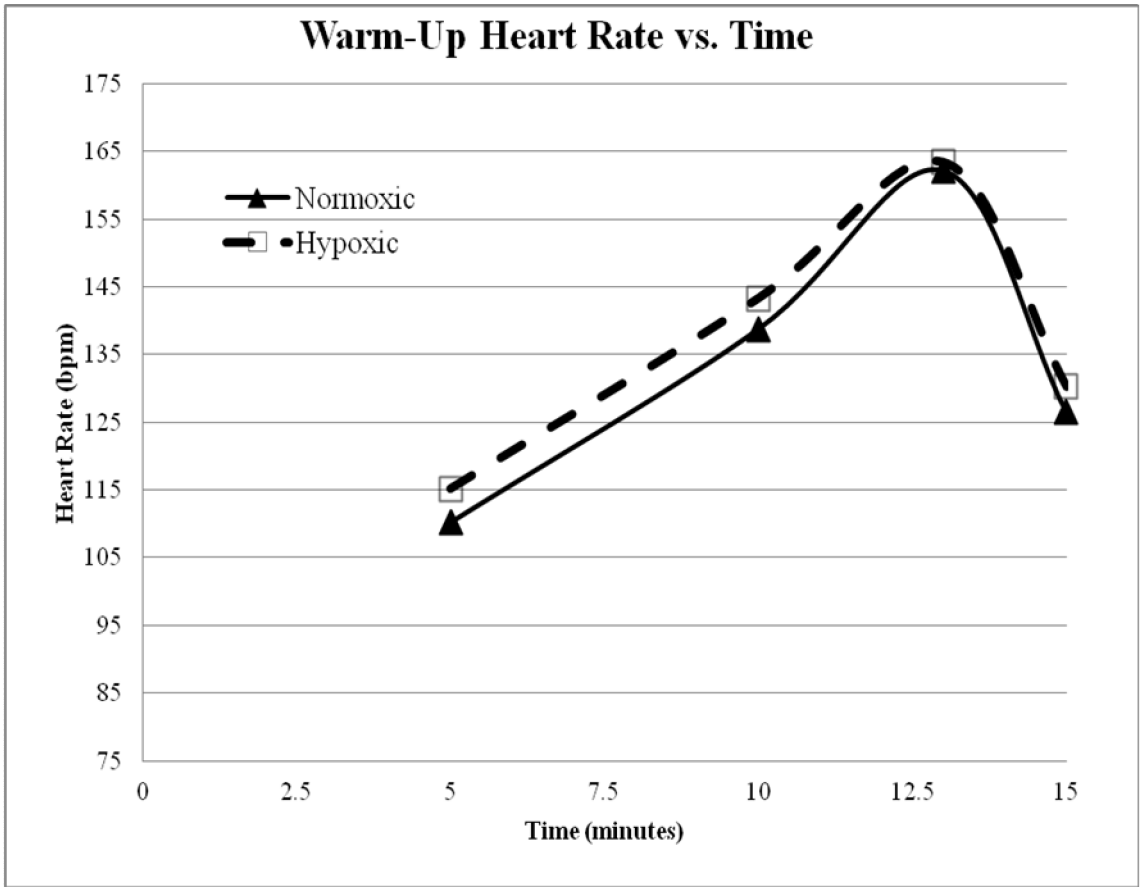

Figure 2. Averaged Heart Rate vs. Time in Warm-Up Conditions

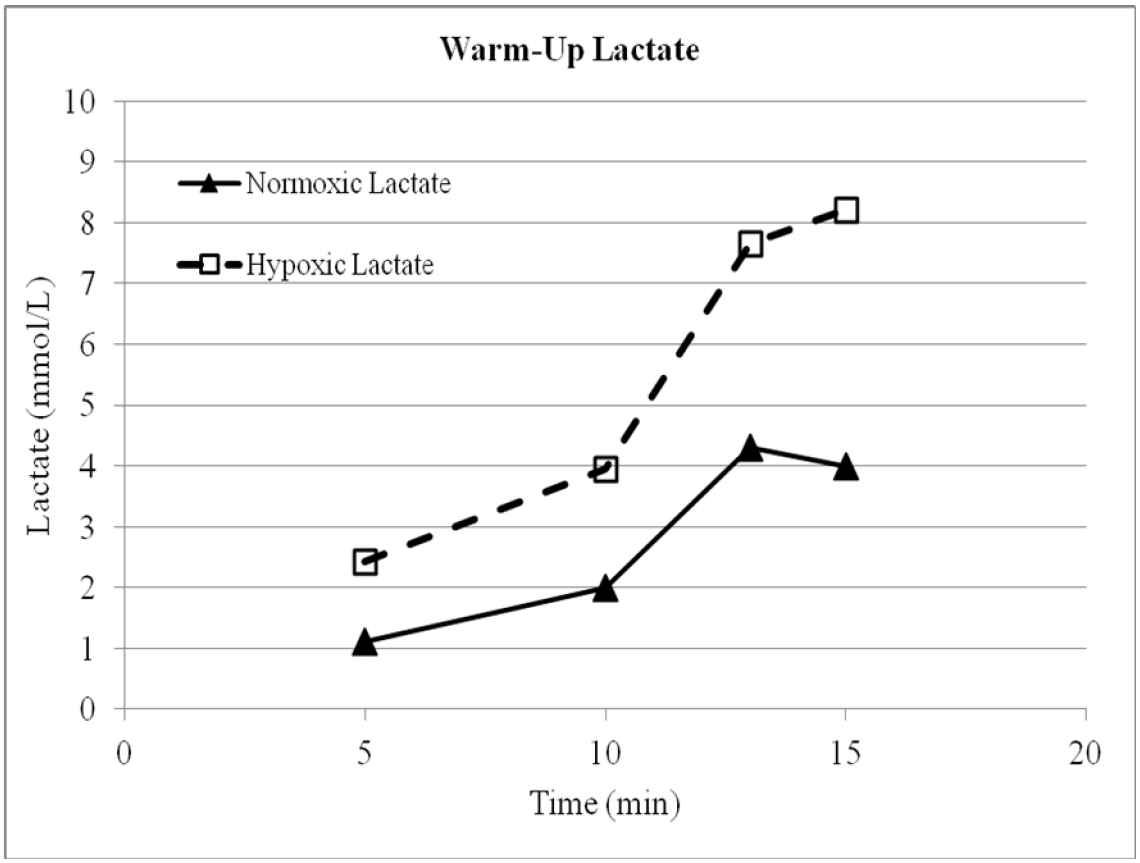

Figure 3. Blood Lactate in Warm-Up Conditions ($p < .05$)*

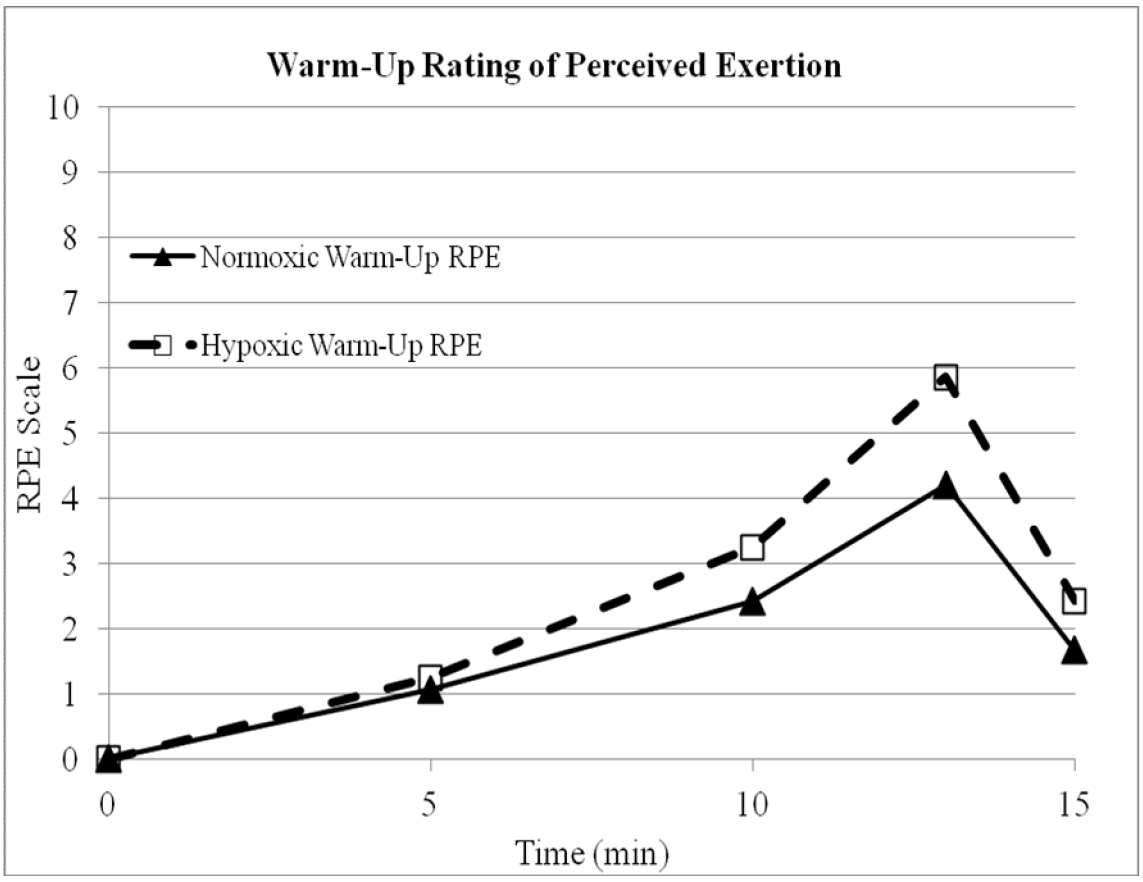

Figure 4. Rate of Perceived Exertion in Warm-Up Conditions ($p < .05$)*

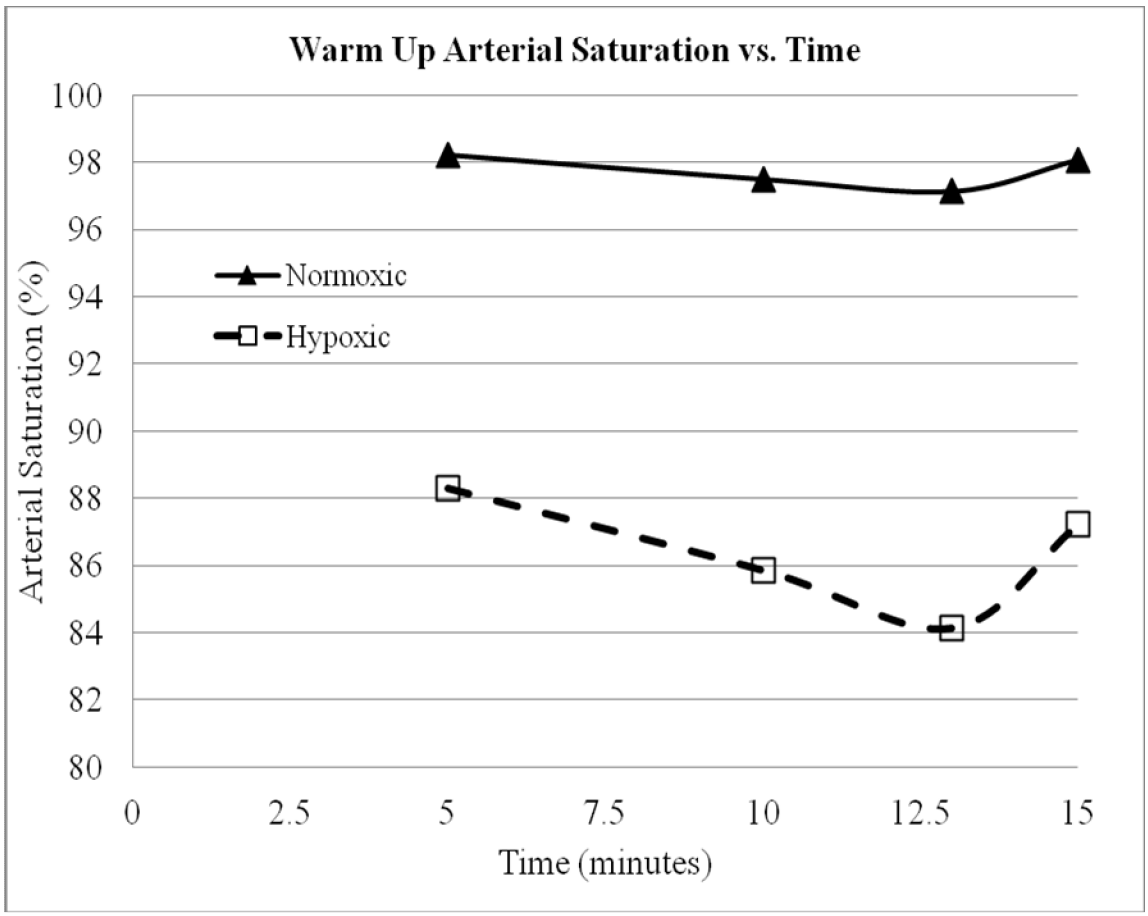

Figure 5. Warm-Up S_aO_2 in Warm-Up Conditions ($p < .05$)*

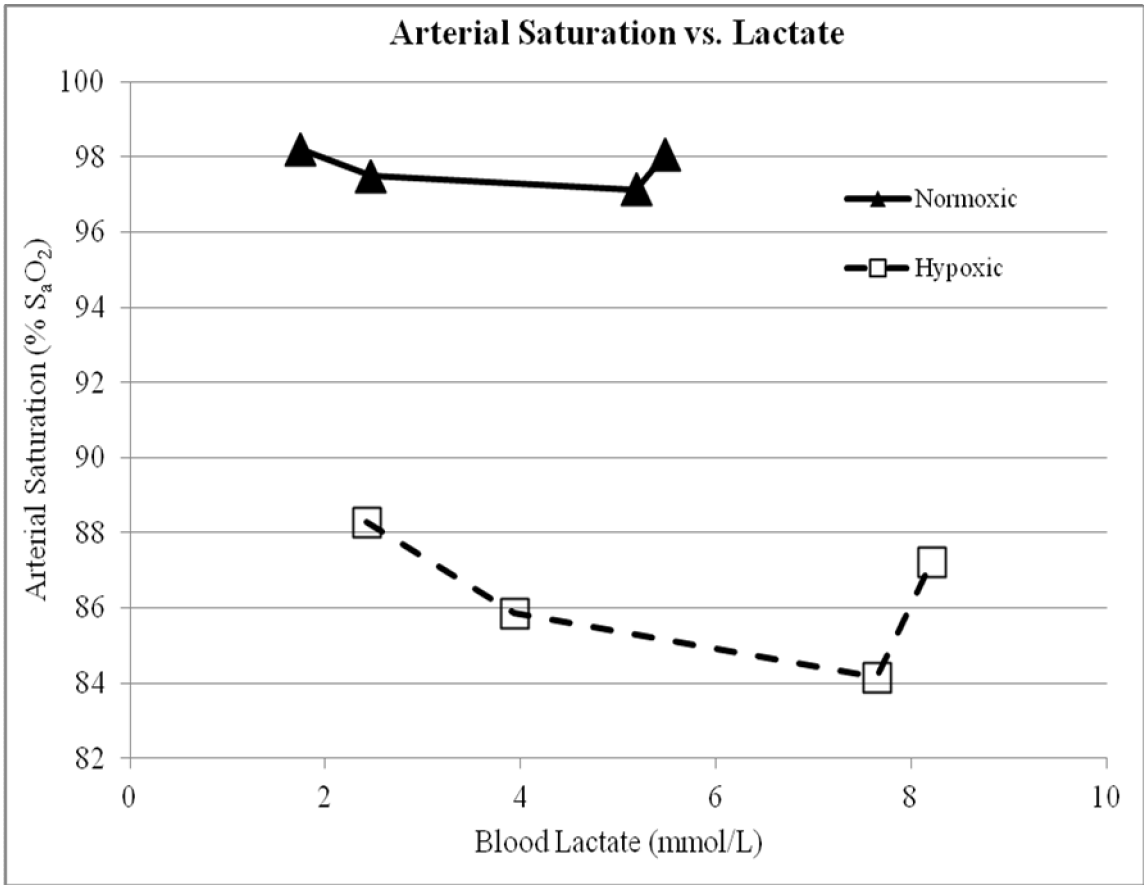

Figure 6. Arterial Saturation vs. Lactate in Warm-up conditions ($p < .05$)*

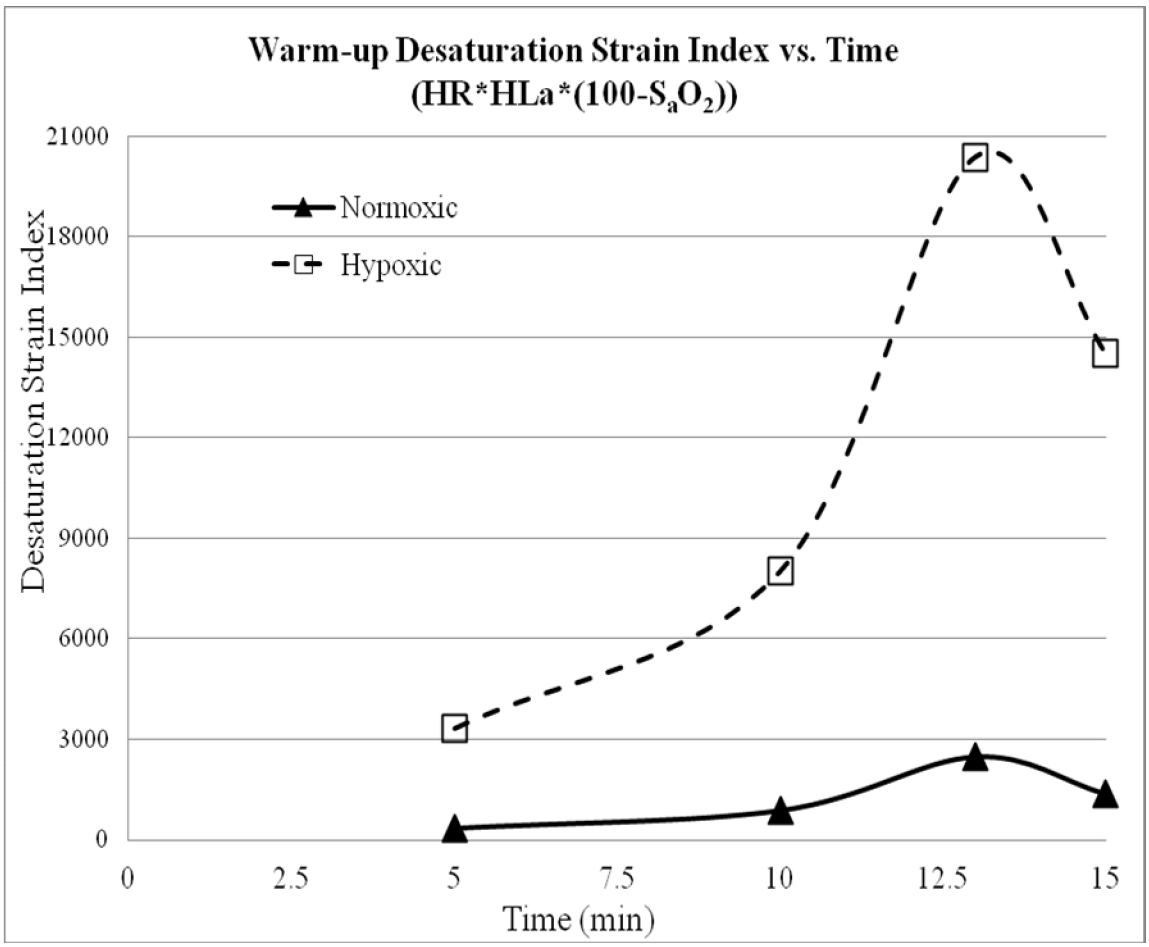

Figure 7. Desaturation Strain Index for Warm-up Conditions ($p < .05$)*
*As an indicator of physiological strain during performance, desaturation strain index is combined effects of HR, HLa, and S_aO_2.

Table 2. Warm-Up Variables of HR, S_aO_2, HLa, and RPE in hypoxic and normoxic condition.

Warm-Up Variable		Time (min)	Mean ± SD
Hypoxia:	HR	5	115.1±18.2
	(bpm)	10	143.2±21.5
		13	163.4±18.7
		15	130.2±13.0
	S_aO_2	5	88.3±2.8
	(%)	10	85.9±2.3
		13	84.1±2.3
		15	87.2±2.9
	HLa	5	2.4±1.0
	(mmol/L)	10	3.9±0.8
		13	7.7±1.9
		15	8.2±2.2
	RPE	5	1.3±0.6
		10	3.3±1.0
		13	6.0±2.2
		15	2.3±0.8
Normoxia:	HR	5	110.2±8.4
	(bpm)	10	138.7±10.8
		13	162.1±8.1
		15	126.5±7.8
	S_aO_2	5	98.2±1.7
	(%)	10	97.5±2.3
		13	97.1±1.6
		15	98.0±1.4
	HLa	5	1.74±0.6
	(mmol/L)	10	2.5±0.5
		13	5.2±1.2
		15	5.5±1.5
	RPE	5	1.0±0.5
		10	2.4±0.8
		13	4.3±1.3
		15	1.5±0.5

Using separate Repeated-Measures ANOVA's, time trial PO, HR, HLa, RPE, and S_aO_2 was compared for each of the 4 conditions: HH, HN, NH, and NN ($p < .05$). Post hoc testing using Fisher's LSD indicated the decrease in PO during hypoxia compared to normoxia to be meaningfully significant (p = 0.000) beginning at the 2.0 km mark (Figure 9), where all 4 combinations of hypoxic (HH and NH) and normoxic (HN and NN) time trials are significantly different from one another. As calculated in the warm-up measurements, the two hypoxic time trials and two normoxic time trials were averaged for comparison between conditions. Heart rate was significantly lower (p = 0.036) with hypoxia at 4-5 km (Figure 10). Time trial RPE was significantly different only at 1 km in NH vs. NN only (p = 0.047), with no significant differences from that point on (Figure 11). Blood lactate was not significantly different throughout the time trial (Figure 12). As expected, there was a significant decrease in S_aO_2 during the hypoxic trials (p = 0.000) (Figure 13).

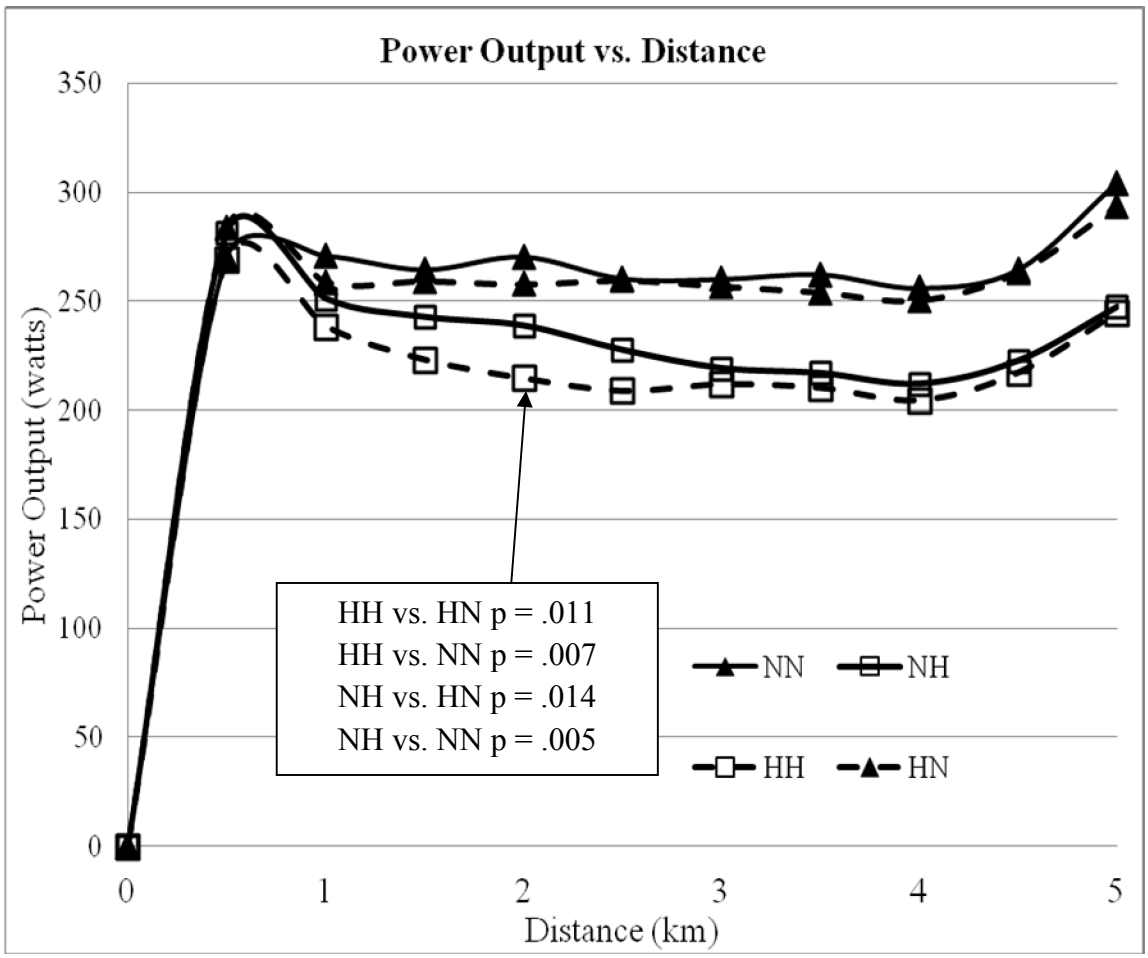

Figure 8. PO for the duration of the time trial for 4 conditions: HH, HN, NH, and HH ($p < .05$ beginning @ 2.0 km until completion)*

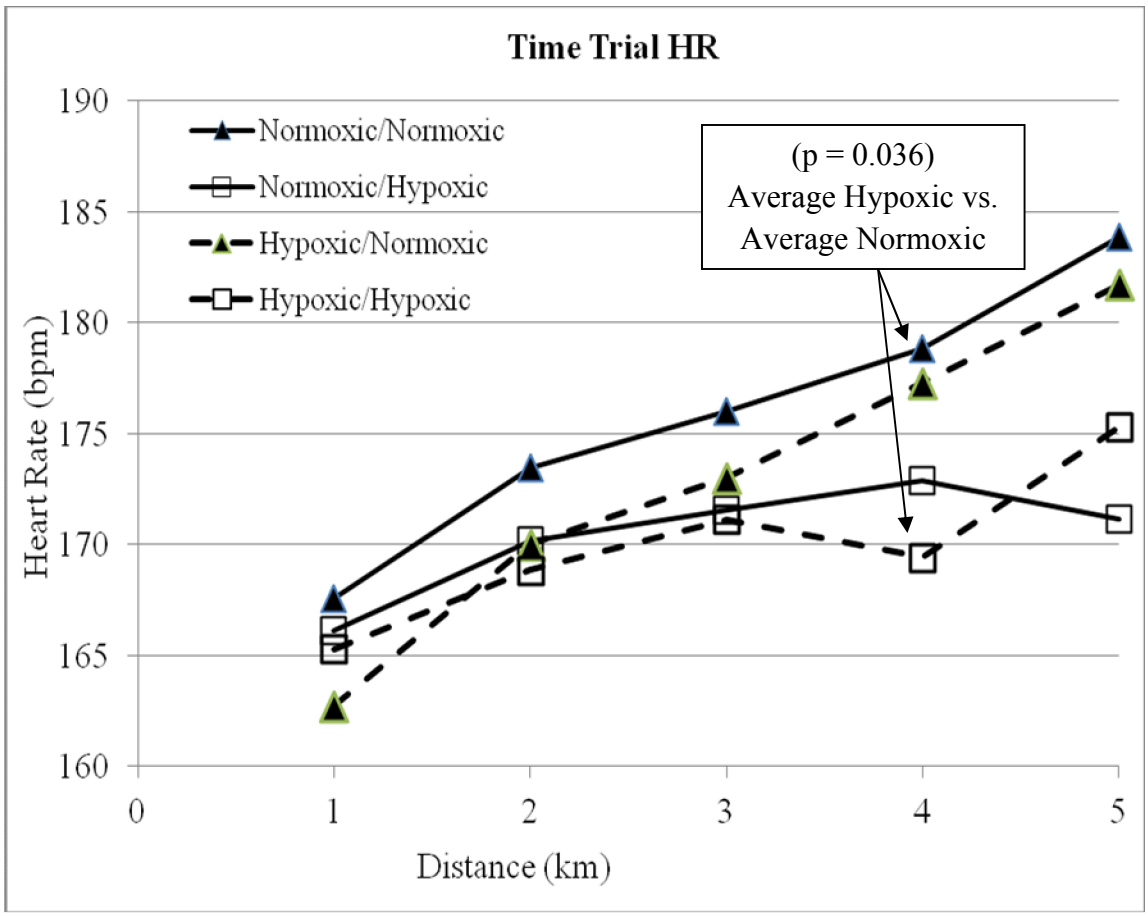

Figure 9. HR for the duration of the time trial for each condition
($p < .05$ @ 4.0 km until completion)*

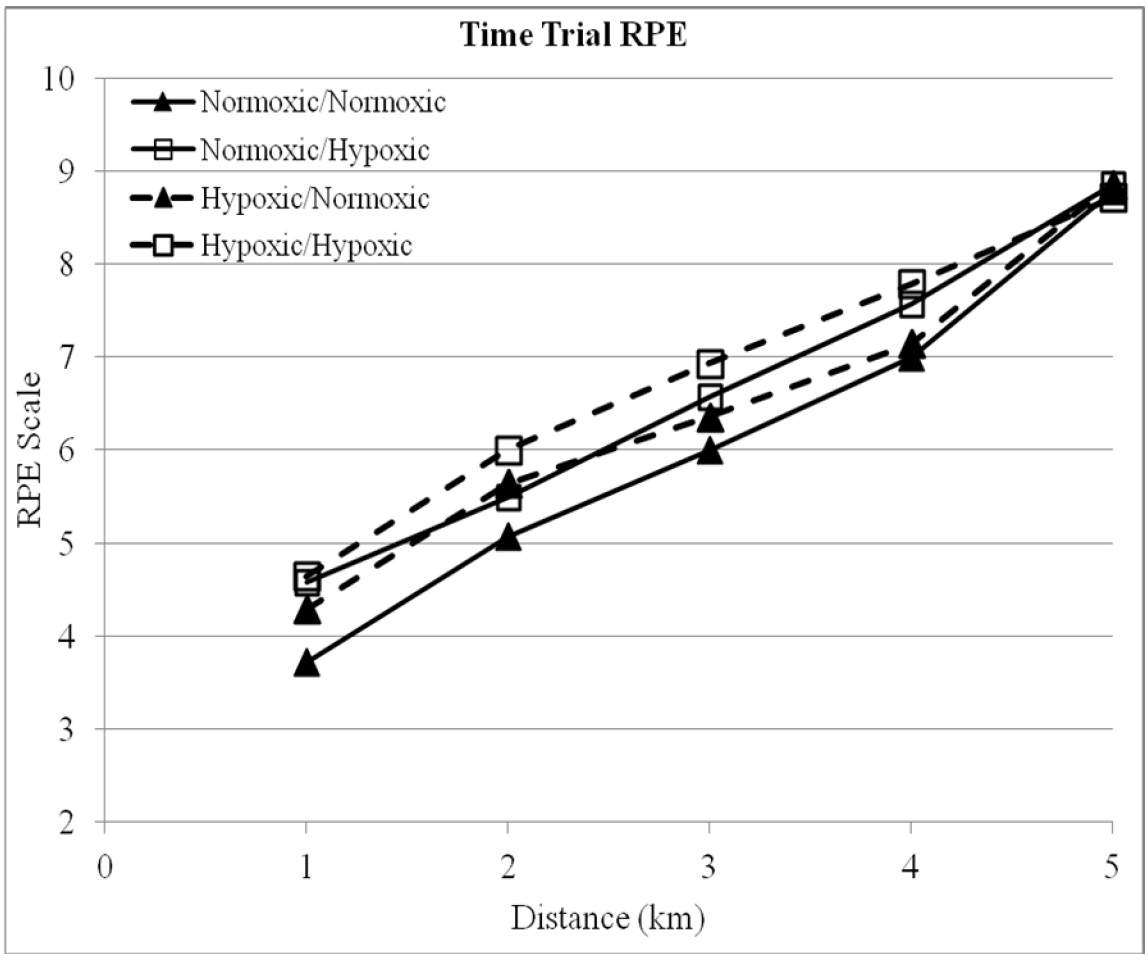

Figure 10. RPE for the duration of the trial for each condition

Figure 11. Blood Lactate for the duration of the trial averaged for hypoxic and normoxic time trial conditions

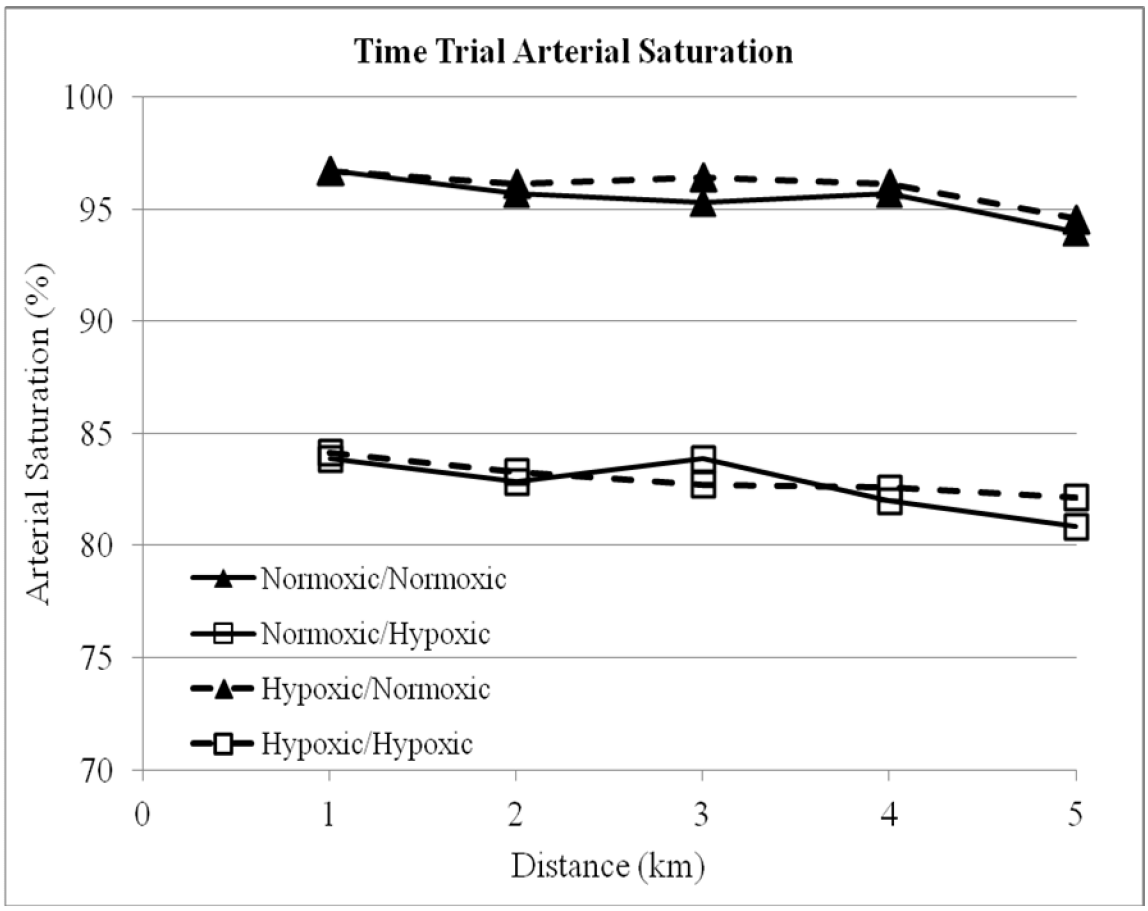

Figure 12. S_aO_2 for the duration of the trial for 4 conditions ($p < .05$)*

Since power output could be individually adjusted during the time trial, significant differences were seen in time to completion with the hypoxic time trials significantly slower than the normoxic trials. Table 3 presents the time, mean PO, and peak PO for each warm-up and time trial condition.

Table 3. Time Trial Results

	NN Mean	SD	NH Mean	SD	HH Mean	SD	HN Mean	SD
Time (min)	8.19	±0.62	8.59	±0.56	8.74	±0.57	8.24	±0.62
Mean PO (watts)	267.57	±51.22	235.00	±41.69	223.71	±37.77	263.43	±51.77
Peak PO (watts)	393.29	±120.09	410.29	±197.23	375.86	±94.45	413.00	±145.43

*NN: Normoxic warm-up/Normoxic time trial, NH: Normoxic warm-up/Hypoxic time trial, HH: Hypoxic warm-up/Hypoxic time trial, HN: Hypoxic warm-up/Normoxic time trial

DISCUSSION

The results of this study demonstrate that, despite large and significant disparities in responses during the normoxic and hypoxic warm-up conditions, PO decreases during hypoxic time trials do not occur until 2 km. In Figure 9 (PO vs. distance) it is visually evident that these trends begin around 1 km, reaching significant differences by 2 km which continue for the duration of the time trial. The magnitude of this decrement in PO agrees with various studies of hypoxic time trial challenges (Amann et al. 2006; Amann et al., 2007b, Calbet et al., 2009a; Johnson et al., 2009; Henslin et al., In Press). Ensuring sufficient tissue oxygenation despite the stress of hypoxia and exercise requires numerous essential physiological and metabolic adjustments (Mazzeo, 2008). Amann et al. concluded that peripheral muscle fatigue reached a critical "threshold" regulating whole body exercise regulated by the central motor drive in a protective manner (2006). With the significant differences in RPE, HLa, and S_aO_2 upon completion of the warm-up, we would have expected initial PO to match this discrepancy. However, starting PO seemed unaffected by the warm-up conditions, implying that the anticipatory template was capable of initially overriding these differences. These results agree with the findings of Henslin et al. (In Press) which found that reductions in power output were not simultaneously decreased with arterial saturation. This suggests the magnitude of the pre-exercise template at the onset of exercise.

Subjects were not blinded to the distance of the time trial and simulation of competitive conditions was intended. This initial similarity in PO supports the concept

Ulmer (1996) termed "teleoanticipation," which involves central regulation with the inclusion of the end point in mind. This model has been supported by numerous studies and review articles investigating the role of a central programmer in paced activity (Hampson et al., 2001; Noakes et al., 2005; St Clair Gibson et al., 2004; St Clair Gibson et al., 2006; Swart et al., 2009). The anticipatory template for exercise plays an important role in the development of pacing strategy, yet other elements of homeostatic maintenance must be considered as well. The results of this study support the idea of the complexity model, which involves multiple feedback and anticipatory mechanisms communicating with the central governor (Hampson et al., 2001; Lambert et al., 2005; Noakes et al., 2005; St Clair Gibson et al., 2004; St Clair Gibson et al., 2006; Swart et al., 2009). The afferent sensory pathways apparently communicate to the brain the physiological changes occurring with exercise, while the efferent pathways in turn adjusts power output to accommodate the limitations of the body (Amann et al., 2007a, Amann et al., 2007b). The present data suggest that it takes between 2 to 3 minutes (~2 km) into the time trial for central motor output adjustments to be made (as manifested in adjustments in PO) from physiological feedback of afferent muscle fibers. As discussed by Noakes et al. (2005), the regulation of pacing is a dynamic, complex feedback system in which the primary goal is to prevent inappropriately large homeostatic changes. This implies that the central programmer directs either the duration of exercise or the power output to end before catastrophe can occur, maintaining a 'protective threshold' for maximal effort (Mauger et al., 2011).

In athletic performance, the extra motivation of competition may drive this threshold closer towards maximum (but never fully reached) for minor performance

improvements which may amount to significant competitive results. Foster et al. (2002) describes this as "a general balance between a pre-exercise template (based on experience with an event) and feedback from a number of receptors, indicating the magnitude of homeostatic disturbance cause by the exercise bout." There is no single factor which causes alterations in pacing strategy, but the combination of numerous anticipatory and sensory factors. During athletic competition, it is essential for the athlete to prepare an effective pacing strategy and constantly be interpreting and responding to environmental and internal cues (Amann, 2011; Foster et al., 2002; Mauger et al., 2011; Tucker et al., 2009). Thus, the initial burst of PO seen at the start of a time trial may signify the complete engagement of the anticipatory template, overriding warm-up effects. In our study, as the time trial progresses the physiological feedback (i.e. HR, HLa, S_aO_2) during the hypoxic condition must further engage the central governor's primary homeostatic mechanisms, in turn causing the decreases in PO. The regulation of motor unit recruitment is determined through calculations of the central programmer to prevent overexertion, which would critically jeopardize homeostasis (Noakes et al., 2001). Studies have evaluated the influence of hypoxia on various physiological mechanisms (Amann et al., 2007a; Amann et al., 2007b; Faoro et al., 2010; Pirkmajer et al., 2010; Richalet et al., 2010; Valli et al., 2011) and include cardiovascular and neuroendocrine responses to exercise (Kjaer et al., 1999) and central governor regulation during hypoxic exercise (Amann et al, 2006; Noakes et al., 2001). In regards to the hypoxic influences on the cardiovascular and physiological aspects of pacing manipulation, our study is in agreement with this research.

In this study decreases were seen in HR during the hypoxic time trials, which seem to coincide with the decreases in PO. It would be logical to expect a significant increase of HR with the environmental stress of the hypoxic time trial, but HR's do not follow this trend. This agrees with other studies on hypoxic exercise performance and heart rate response (Calbet et al., 2009b; Wagner et al., 2000). Wagner et al. concluded that the blunted heart rate response seen with altitude is related to that of exercise intensity versus some kind of pathophysiological response (2000). The supposed protective mechanisms of regulation of PO to prevent a catastrophic endpoint may also be the same controlling factors of HR response to hypoxic exercise (Benoit et al., 2003; Calbet et al., 2009a; Mollard et al., 2007).

An interesting finding of this study is that no change occurred in blood lactate (HLa) between the time trial conditions. This brings to mind the "lactate paradox" as discussed in terms of exercising in altitude (Noakes et al., 2009; Wagner & Lundby, 2007). Pacing can be described as "a strategy employed to avoid catastrophic failure in any peripheral physiological system" (St Clair Gibson et al., 2006). Foster et al. (2002) suggests that athletes accept a certain amount of 'calculated risk' by inducing larger homeostatic disturbances than previously encountered in an attempt to improve. Since the working muscle has a certain ability to accumulate lactate during exercise, we see a significant separation between the hypoxic and normoxic warm-up conditions. However, as the subject enters into the time trial this accumulation may be approaching maximum levels, and therefore HLa production may be limited as a protective mechanism during hypoxia. This idea is in agreement with Kayser et al. (1994) who noted that ". . . during chronic hypobaric hypoxia, the central nervous system may play a primary role in

limiting exhaustive exercise and maximum accumulation of La (lactate) in blood." Although no variations occurred between conditions, all trials elicited HLa levels well above the 8-9 mmol criteria for maximum efforts.

The complexity of this experimental design provided several challenges for creating an optimal competitive environment. Because of the design of the air concentration chambers, the switching of F_iO_2 was not visible to the subject. Investigators performed the same actions regardless of the warm-up or time trial F_iO_2 conditions to prevent any bias from the subjects. Added precautions from the sound of the hypoxic air leaving the tank may be beneficial for future studies. Although none of the subjects mentioned the sound as a determining factor of their performance, the investigators noticed that it may have been audible near the subject. Future research may be beneficial in comparing the responses of PO, HR, HLa, and RPE with prolonged time trials or varying forms of warm-up conditions or treatments. The conclusions of this study seem to support existing research in the areas of hypoxia and pacing performance.

The question of this study was in regards to manipulation of the feedback responses of exercise. The evidence of peripheral feedback mechanisms is supported in this study, with alterations of PO occurring with varying F_iO_2 concentrations. This indicates that a central programmer is responding to the hypoxic challenge and adjusting motor output accordingly to maintain performance while minimizing endangerment of homeostatic mechanisms. As we have manipulated factors within the time trial cycling performance, we see that central control alterations through afferent feedback occur at approximately 2 km.

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APPENDIX A

INFORMED CONSENT

INFORMED CONSENT

EFFECT OF DISPARITIES OF FEEDBACK ON PACING IN CYCLE TIME TRIALS

I, _____, give informed consent to participate in a research study being conducted at the University of Wisconsin-La Crosse, in the Human Performance Laboratory. The purpose of this study is to test and examine the effect of a low oxygen mixture during warm-up and/or performance on pacing strategy in a cycle time trial. Kate Nyberg, a graduate student in the Clinical Exercise Physiology program, is conducting this study with the supervision of Dr. Carl Foster, a Professor from the Department of Exercise and Sport Science at the University of Wisconsin-La Crosse, and Chris Dodge, the Human Performance Laboratory Supervisor. I give consent to the presentation, publication, and other releases of summary data from the study, which are not identifiable with myself.

I have been informed that my participation in this study will require me to:

a) Perform one maximal exercise test to determine my maximal power output and six cycle time trials on an electronically braked racing bicycle. Each session will take approximately one hour to complete
b) Wear a chest strap heart rate monitor
c) Wear a pulse oximeter, which is similar to a clip attached to my finger
d) Breathe through a scuba-type mouthpiece and wear a nose clip so that my exhaled air may be analyzed and recorded
e) Breathe from a large bag, which may contain room air or air with a low oxygen concentration (hypoxia) (equal to 7000 ft altitude) during the time trials. I will now know the content of the composition of the air prior to each session

I have been informed that there are no risks associated with this study other than fatigue, leg tiredness, and shortness of breath, all of which are similar to participating in a bicycle race. The risk of serious or life-threatening complications is very low (<1/10,000 tests). The test will be stopped immediately upon any complications and there will be persons certified in ACLS and an AED available for every test.

I have been informed that upon my completion of all study requirements I will receive $50 compensation. If I do not complete all study requirements, I will not be eligible to receive the $50 compensation. I will also receive information about my current fitness level. This study will assist scientists to better understand how people pace themselves during strenuous physical activity with altered environmental variables.

I have been informed that the investigator will answer all questions regarding the procedures or any concerns I may have throughout the study.

I have been informed that I am free to decline to participate or to withdraw from the study at any time without penalty.

If I have any questions that may arise, I may feel free to contact Kate Nyberg, the principal investigator, at (763) 486 – 2913, or her supervisor, Dr. Carl Foster, at (608) 785 – 8687. Questions in regards to the protection of human subjects may be addressed to the University of Wisconsin-La Crosse Review Board for the Protection of Human Subjects at (608) 785 – 8124.

Subject:_____ Date:_____

Investigator:_____ Date:_____

APPENDIX B

REVIEW OF LITERATURE

The purpose of this paper is to review the literature regarding the pre-exercise template and physiological feedback mechanisms of pacing strategies. We will manipulate these factors with cycling time trial performances in hypoxic environmental conditions.

Introduction

Pacing can be described as "a strategy employed to avoid catastrophic failure in any peripheral physiological system" (St Clair Gibson et al., 2006). There has been extensive interest in the area of pacing and the peripheral and central physiological mechanisms which control it. Athletes utilize pacing strategies for both competition and performance enhancement, and numerous practice hours are spent perfecting pacing strategy. Foster et al. (2002) suggests that athletes accept a certain amount of 'calculated risk' by inducing larger homeostatic disturbances than previously encountered in an attempt to improve. During athletic competition, it is essential for the athlete to prepare an effective pacing strategy and constantly be interpreting and responding to environmental and internal cues (Foster et al., 2002; Mauger et al., 2011; Tucker et al., 2009).

These strategies may also be described as a natural phenomenon of life and applicable to the overall functioning of our species, not unique to athletic competition (Noakes et al., 2005). Over the years, research examining this concept has included measurement parameters such as heart rate (HR), electromyography (EMG), power output (PO), and rating of perceived exertion (RPE) (Albertus et al., 2005; Ansley et al.,

2004; Foster et al., 2003; Rauch et al., 2005; St Clair Gibson et al., 2001). Several models of pacing strategy have been presented, including models of both central and peripheral fatigue within a paced activity.

Numerous studies have been conducted to examine the various factors of pacing strategy, manipulating such external factors as oxygen concentration and temperature (Amann et al., 2007; Henslin et al., In Press; Johnson et al., 2009; Kjaer et al., 1999; Noakes et al., 2001). These studies examined the response of parameters such as power output and RPE with changing environmental conditions. Other studies have researched the effects of deceptive feedback, such as incorrect distance feedback or unknown time trial distance (Albertus et al., 2005; Ansley et al., 2004; Mauger et al., 2009; Mauger et al., 2011). The responding pacing strategy was observed to test the influences of the pre-exercise template and previous experience. The purpose of this study is to combine the objectives of these studies by observing the adapted pacing strategy when physiological feedback (using hypoxic environmental conditions) is altered between warm-up and time trial performance (altering the athlete's anticipatory template).

Feedback and Pacing Strategy

Foster et al. (2002) describes pacing strategy as "the process whereby humans regulate their rate of energy expenditure in a way that allows them to complete a task in the minimal time, while controlling the magnitude of homeostatic disturbance." The various pacing strategies have been developed for performing at a high enough level to optimize workload while preventing detrimental effects (Foster et al., 2002; Foster et al., 2003; Noakes et al., 2005; Rauch et al., 2005; St Clair Gibson et al., 2005; Swart et al., 2009).

A study conducted by Ansley et al. (2004) researched the anticipatory factors influencing pacing strategy through deceptive distance feedback. Eight healthy males performed 6 Wingate anaerobic tests, yet the duration for 4 of the trials was actually longer than what the athlete was told prior to the trial. The results of the deception trials showed significant differences in power output in the last seconds of the performance, indicating that the athlete had a pre-programmed endpoint in mind prior to beginning the session. Wingate anaerobic tests elicit large amounts of skeletal muscle fatigue, yet this is not the only factor which regulates pacing. The anticipated amount of time the athlete was expecting as well as what they have previously experienced during a Wingate test played main roles in determining power output during the first 30 seconds (the typical amount of time for test completion). After this point power output began to decline, even though the athlete was unaware of the increased duration of the trial. This implies that central regulation is involved in "pre-setting" an exercise bout prior to the activity.

Albertus et al. (2005) conducted another study with inaccurate distance feedback researching pacing strategies. Fifteen well-trained cyclists performed a peak power

output test, familiarization test, and four 20-km cycling time trials where they received only distance feedback in 1-km distance splits. When incorrect distance feedback was given to the athletes, pacing strategies were unaltered. This agrees with the previous study in that the pace seems to be set prior to the time trial, regardless of the external distance feedback provided. The researchers concluded that "such regulation would occur in an anticipatory manner based on expectations of exercise duration, before commencement of the trial."

Expanding on this idea is another study completed by Mauger et al. (2009) determining the effects of distance feedback. Random assignment of eighteen well-trained male cyclists into a control or experimental group performed 4 consecutive 4-km time trials. The control group received prior knowledge of the distance to complete as well as distance feedback during each trial, while the experimental group only knew that all trials were of the same distance. Over the trials, the experimental group made successive time improvements. The researchers concluded that the initial trials for the experimental group served as their previous experience which they based the pacing of the remaining trials off of, in spite of the unknown distance. Researchers also discussed the idea of working backward to develop an effective pacing strategy, such that the experimental group developed their pace based off the endpoint.

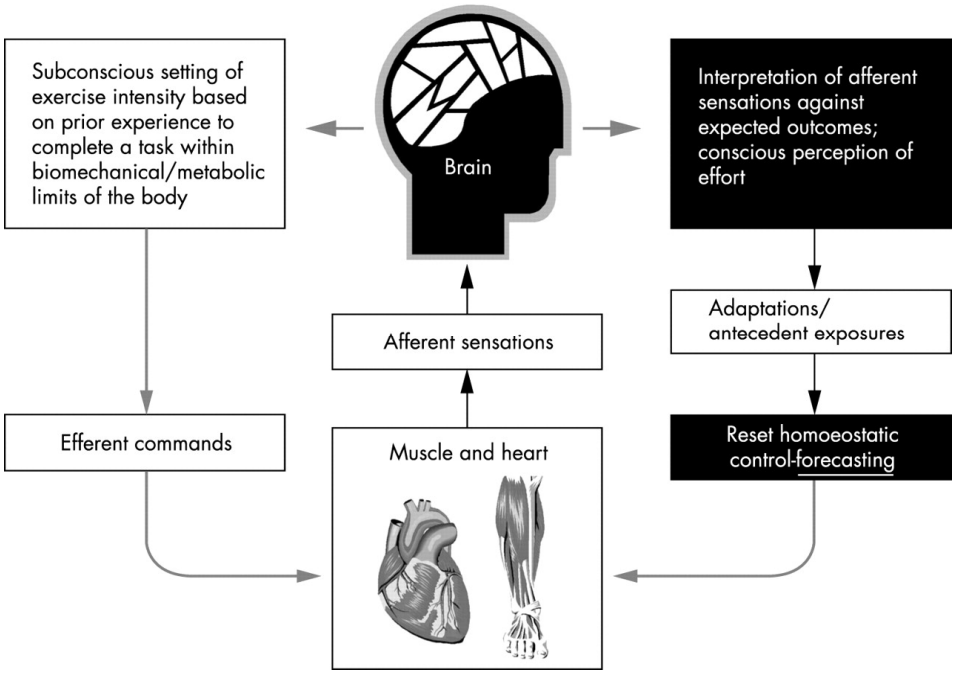
These studies all correlate with a concept of Ulmer (1996) termed "teleoanticipation," which involves central regulation with the inclusion of the end point in mind. This model has been supported by numerous studies and review articles investigating the role of a central programmer in paced activity (Hampson et al., 2001; Noakes et al., 2005; St Clair Gibson et al., 2004; Swart et al., 2009). Ulmer (1996)

explains that “anticipation concerns not only the basis of harmonic, biomechanically optimized motion, but also a teleoanticipation for the optimal arrangement of exertion, which avoids early exhaustion before reaching a finish point.” The anticipatory template for exercise plays an important role in the development of pacing strategy, yet other elements of homeostatic maintenance must be considered as well.

Central and Peripheral Feedback

In an effort to maintain bodily homeostasis during pacing, the models of catastrophe and complexity have both been presented. Originally, the model of catastrophe, i.e. the idea that exercise ceased as a result of metabolic byproduct build-up or complete motor unit recruitment of the skeletal muscle, was accepted (Noakes et al., 2005). With more research scientists realized that paced activities were much more intricate than this resulting in the development of the complexity model, which involves multiple feedback and anticipatory mechanisms communicating with the central governor (Hampson et al., 2001; Lambert et al., 2005; Noakes et al., 2005; St Clair Gibson et al., 2004; St Clair Gibson et al., 2006; Swart et al., 2009). As previously mentioned, the concept of teleoanticipation has been a central model for paced activity (Ulmer, 1996). Ulmer describes feedback system control as a process of communication between the central nervous system (CNS) and the working skeletal muscle via afferent and efferent control. The afferent sensory pathways communicate to the brain the physiological changes occurring with exercise, while the efferent pathways in turn adjusts power output to accommodate the limitations of the body.

A review article by Lambert et al. (2005) investigates this further and provides this diagram as an explanation of the complex systems model of fatigue:



This model describes the multiple aspects of physiological and psychological integration involved in pacing strategy development. Lambert et al. (2005) explains the teleoanticipation model as "both feed forward planning and feedback control from afferent changes associated with peripheral metabolic structures and the external environment, and also incorporates knowledge acquired from prior exercise bouts." The role of central governor control has been confirmed in various studies measuring power output (PO), electromyogram (EMG), and muscle glycogen (Hug et al., 2003; Rauch et al., 2005; St Clair Gibson et al., 2001; St. Clair Gibson et al., 2005; Swart et al., 2009).

One study by Swart et al. (2009) established CNS regulation in pacing by experimenting with a centrally acting stimulant. Eight elite cyclists ingested 10 mg

methylphenidate in a randomized, placebo-controlled crossover trial. The results showed subjects receiving the tested variable had significantly higher PO, oxygen consumption, HR, blood lactate concentration, and ventilator volumes while EMG activity remained unchanged. This would support the models of a central governor, since the catastrophe model would assume that EMG activity would increase sequentially with other factors.

A study by Amann et al. (2006) examined the interaction of centrail and peripheral control with the resulting effects on power output. Eight trained males performed four 5 km cycling time trials with varying amounts of arterial saturation contents. Through their study they concluded that performance was determined "to a significant extent by the regulation of central motor output to the working muscle in order that peripheral muscle fatigue does not exceed a critical threshold."

As discussed in the review by Noakes et al. (2005), the regulation of pacing is a dynamic, complex feedback system in which the primary goal is to prevent significant homeostatic changes. This implies that the central programmer directs the duration of exercise to end before catastrophe can occur, maintaining a 'protective threshold' for maximal effort (Mauger et al., 2011). In athletic performance, the extra motivation of competition may drive this threshold closer towards maximum (but never fully reached) for minor improvements which could amount to significant results. Foster et al. (2002) describes this as "a general balance between a pre-exercise template (based on experience with an event) and feedback from a number of receptors, indicating the magnitude of homeostatic disturbance cause by the exercise bout." There is no single factor which causes alterations in pacing strategy, but the combination of numerous anticipatory and sensory factors. Swart et al. (2009) describes peripheral fatigue as "either the excessive

accumulation or depletion of key chemicals which interferes with cross bridge cycling in the exercising muscles, impairing their capacity to produce force." Central fatigue "proposes that related chemical changes in the brain alter cerebral function, reducing the ability to maintain central motor drive to the exercising muscles." One tool researchers have utilized in analyzing fatigue and the central programmer is the rating of perceived exertion (Crewe et al., 2008; Gearhart et al., 2005; Hampson et al., 2001; Henslin et al., In Press; Johnson et al., 2009; Foster et al., 2002; Swart et al., in press; Tucker et al., 2009). It has been proposed that the sensation of fatigue is the initial protective mechanism of the brain to prevent exercise from reaching detrimental levels (Noakes et al., 2005).

Pacing and Fatigue

There have been various concepts presented for the relationship between ratings of perceived exertion and exercise fatigue. Tucker et al. (2009) proposed a conceptual model for "how the RPE mediates feedforward, anticipatory regulation of exercise performance" for application to research studies manipulating pacing strategies.

A study by Crewe et al. (2008) suggests that there is a linear relationship between exercise duration and RPE increases; therefore it would be possible to predict the termination of exercise very early into the performance. Seven subjects performed five cycling trials in an environmental chamber. At the temperature of 15 degrees Celsius trials were performed at intensities of 55, 60, and 65% and at 35 degrees Celsius trials were performed at intensities of 65 and 70%. RPE rose in a linear fashion throughout the trials, and researchers concluded that RPE could be used as a prediction tool. They suggest that "the subconscious brain must be able to forecast the duration of exercise and then set the rate of increase in RPE at a greater level… so volitional fatigue occurs before, in this case, the body temperature can rise excessively." This would mean that the brain can 'sense' these changes and may create a 'pre-set' RPE based on the initial exercise factors.

Another study by Gearhart et al. (2005) examines the relationship between power output and ratings of perceived fatigue. This study found that higher exertion ratings were felt with greater resistance versus higher pedal rates at similar workloads. The increased resistance would cause a higher overall force developed by the skeletal muscle, implying that the amount of muscular tension mediates the amount of exertion felt by the athlete.

Yet another idea presented by Swart et al. (In Press) expands on the dissociation between sensations of exercise and awareness of effort. They proposed a new measure of task effort called the Task Effort and Awareness (TEA) score in an effort to distinguish from the ratings of perceived exertion (RPE). Well-trained cyclists performed several maximal and submaximal trials, and were able to clearly distinguish between their feelings of physical effort and psychological effort. The overall sensation of effort and fatigue may then be a combination of both feed-forward and adaptive mechanisms. This provides further evidence of the central programmer involvement in the conscious maintenance of controlling homeostasis.

Hampson et al. (2001) summarizes that perception of effort may be regulated by "an integration of multiple afferent signals." Depending on the particular mode of exercise performed, a certain physiological signal may become dominant in triggering RPE increases. This model also includes central teleoanticipation as discussed by Ulmer (1996) in that RPE is meant to terminate activity before the metabolic and/or mechanical upper limit is met. As scientists have worked to distinguish the various factors affecting RPE and pacing strategy, various forms of manipulation have been tested.

Pace Manipulation

As mentioned previously, numerous studies have manipulated the variables of pacing strategy (Albertus et al., 2005; Amann et al., 2007; Ansley et al., 2004; Henslin et al., In Press; Johnson et al., 2009; Kjaer et al., 1999; Mauger et al., 2009; Mauger et al., 2011; Noakes et al., 2001). One well known study by Rauch et al. (2005) manipulated the amount of muscle glycogen content during a paced activity. In this research eight well-trained endurance cyclists completed 2 hours of cycling at ~73% of maximum oxygen consumption, including five sprints at 100% of peak sustained power output every twenty minutes, followed immediately by a one hour time trial. The same performances were completed once when subjects were carbohydrate loaded and once when they were non-loaded and the ending result of muscle glycogen concentrations was basically identical. Despite this, carbohydrate loading improved overall power output and utilization of muscle glycogen during the one hour trial in comparison to the trial without. Researchers concluded that subjects "may have paced themselves according to internal physiological feedback, informing them of the maximum work rate that they would be able to sustain for 1 hour without developing premature fatigue." A remarkable finding of this study is that the adjustment of pacing strategy was actually made within the first 1-2 minutes of time trial. This shows the bodies responsive capabilities and efficiency of interpreting sensory input information. The theory of central nervous system adjustments according to muscle glycogen concentration was made in this study, suggesting that this may be one of the "critical" end point concentrations that is monitored through the central governor by chemoreceptors in the muscle (termed "glycostat").

Another external manipulator is that of pacing strategy and extreme temperature change. Levels et al. (in press) analyzed this through sudden, extreme heat exposure during cycling time trials. These findings were of interest since no change in performance or pacing patterns were made with the sudden onset of temperature change. Thirteen well-trained male subjects performed three 7.5-km time trials in 15 degrees Celsius and 50% relative humidity. The three experimental conditions were: PRECOOL (whole body precooling before exercise and heat stress during time trial), HEAT (no precooling, heat stress during time trial), and CONTROL (no precooling and no heat stress). Heat stress was applied by a panel of infrared heaters that was quickly positioned in front of the cycle after 1.5 km and quickly removed after 6 km. A combination of whole-body precooling and extreme heat exposure decreased average power output during the cycling time trial. In this study it appears as though extreme temperature changes had little effect on performance and pacing, which may imply that this factor serves as a weak signal to overall pacing modification. It is worth mentioning that one possibility for this minimal change is the limited rise in core temperature, which may not have had adequate time during the trial to rise to levels of significance.

As discussed previously, another experiment analyzing temperature manipulation is that of Crewe et al. (2008). Seven subjects performed five cycling trials in an environmental chamber. At the temperature of 15 degrees Celsius trials were performed at intensities of 55, 60, and 65% and at 35 degrees Celsius trials were performed at intensities of 65 and 70%. Unlike the results of Levels et al. (in press), the researchers concluded that the hot environmental temperatures did have an effect on performance, based on duration and rate of increase of RPE. They proposed that temperature and

intensity sensors triggered the brain for the determination of exercise duration. The overall experimental data available from pacing strategy manipulation is not conclusive. Another form of environmental manipulation which has been researched extensively is that of hypoxia and its effects on pacing strategy and performance.

Hypoxia and Pacing

Hypoxia may be particularly valuable as a model to evaluate elements of pacing strategy since inspired gas concentrations can be manipulated while the subject is blinded to changes that are being made. Other variables of external manipulation (such as muscle glycogen and temperature alteration) are useful for evaluating feedback mechanisms of the body, yet subjects are aware always aware of the changes occurring. With changes in oxygen concentration, subjects cannot distinguish this change until their physiological feedback mechanisms are engaged. In a review by Noakes et al. (2001), the conclusion was made that the brain has the ability to sense acute hypoxia and to increase blood flow in response. This article further describes that the regulation of motor unit recruitment is determined through calculations of the central programmer to prevent overexertion, which would jeopardize oxygen delivery to vital organs. Studies have evaluated the influence of hypoxia on various physiological mechanisms (Amann et al., 2007; Faoro et al., 2010; Pirkmajer et al., 2010; Richalet et al., 2010) and include cardiovascular and neuroendocrine responses to exercise (Kjaer et al., 1999) and central governor regulation during hypoxic exercise (Noakes et al., 2001). One unique factor of exercise noted in the study by Amann et al. (2007) is the inspiratory muscle work and its effect on peripheral locomotor muscle fatigue. This would result in added peripheral muscular workload, which in turn effects the perception of effort during exercise.

The study by Kjaer et al. (1999) investigated hypoxic exercise effects with epidural anesthesia. Researchers found that time to exhaustion at extreme hypoxia, circulatory responses, concentrations of neuroendocrine hormones, and extramuscular substrate mobilization were not diminished by epidural anesthesia. Because of this, the

conclusion was made that neural feedback was not the primary mediator of adaptations to hypoxia. Instead, the summary was made that "blood-mediated afferent signaling from skeletal muscle to higher circulatory and endocrine centers when exercise is performed in a hypoxic environment" is a more primary component than neural afferent impulses.

Noakes et al. (2001) approaches this in a different way concluding that regulation of exercise does involve central governor regulation. It is proposed that the central governor will sense the acute hypoxic environment and adjust the amounts of muscle mass recruited for exercise to prevent oxygen depletion in the vital organs of the body. This hypothesis states that "skeletal muscle recruitment during severe exercise is regulated by a central governor specifically to prevent the development of progressive arterial desaturation leading to myocardial ischemia or cerebral hypoxia."

This leads into another study by Johnson et al. (2009) investigating arterial hypoxemia and power output (PO) alterations with hypoxic exercise. Ten well-trained cyclists performed randomly ordered 5-km time trials. Subjects began each trial breathing room air and switched (blindly) to hypoxic (HYPOXIC, $F_IO_2 = 0.15$) or room (CONTROL, $F_IO_2 = 0.21$) air at 2 km, then to room air again at 4 km. Results of this study showed a correlation between modifications in power output and the development of arterial hypoxemia, beginning at ~30 seconds into the time trials. The experimental treatment induced large changes in S_aO_2 and found significant PO changes. Researchers summarized that arterial desaturation may be an important, primary signaling mechanism to the central governor to adjust PO accordingly to accommodate to the hypoxic conditions. The presence of central control may be engaged through arterial chemoreceptors. This information correlates with other studies discussing the idea that

the brain and central nervous system (central governor) are dynamically in back and forth communication through afferent sensory pathways (Foster et al., 2003; Lambert et al., 2005; St Clair Gibson & Noakes, 2004; St Clair Gibson et al., 2006).

A recent study by Henslin et al. (In Press) suggested that arterial desaturation immediately prior to a time trial does not change the pattern of power output (PO) at the beginning of a time trial. Eight trained cyclists performed three randomly ordered 3-km time trials while breathing room air (CONTROL) or a hypoxic gas mixture that was either administered before the start of the time trial (EARLY) or at the beginning of the time trial (LATE). In EARLY, hypoxic air ($F_IO_2 = 0.15$) was administered 3 minutes prior to the start of the time trial; the LATE time trial began at the time the subject first breathed hypoxic air. These results call into question the findings of Johnson et al. (2009) that arterial desaturation is a direct controller of power output. These results suggest the importance of the pre-exercise template (which was not manipulated in the study of Henslin et al., In Press) and the importance of changes in metabolic status which occur secondary to limitations in central oxygen transport following administration of hypoxia.

Conclusion

The exact mechanisms behind the central programmer, afferent sensory pathways, and efferent response pathways are not clearly understood. The purpose of this study is to further understand the components of pacing, that is, the relationship between anticipatory factors, previous experience, and physiological feedback mechanisms. Our question is in regards to the manipulation of the feedback template of exercise. If we alter an athlete's pre-exercise template by manipulating warm-up conditions and he or she approaches performance with a certain pacing strategy, we will observe how he or she responds when his or her physiological feedback contradicts the anticipatory template. We will measure these changes through hypoxic exercise, hypothesizing that when F_IO_2 changes from that experienced during the warm-up, PO will adapt to match the PO normally experienced (indicating the response of the central programmer to afferent information). The timing of this adaptation will be in the first 1-2 minutes of exercise.

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EFFECT OF DISPARITIES OF FEEDBACK ON PACING IN CYCLE TIME TRIALS

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We recommend acceptance of this thesis in partial fulfillment of the candidate's requirements for the degree of Master of Science in Clinical Exercise Physiology

The candidate has completed the oral defense of the thesis.

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